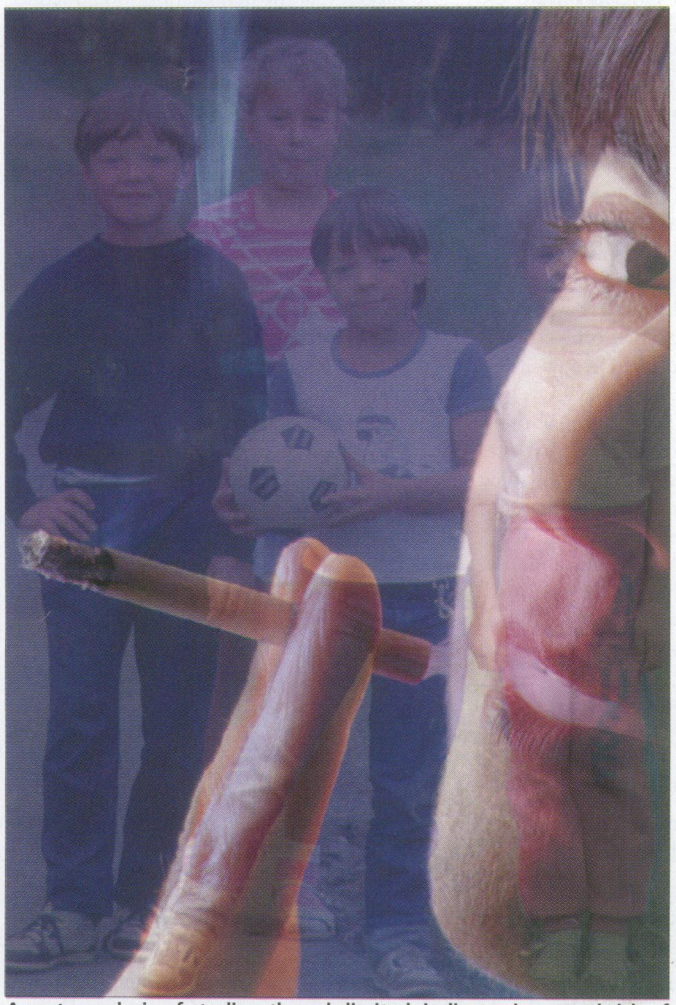
Sins of the Father

Parental Smoking and Childhood Cancer

Exposure to passive (or environmental) tobacco smoke has been implicated in numerous childhood ailments ranging from low birth weight to middle ear disease. Passive smoke has also been suspected, but not proven, to increase the risk of childhood cancer. Clarifying this issue is crucial not only for the protection of children's health but also for the fashioning of policy concerning smoking in public and private facilities. In this month's issue, Paolo Boffetta and colleagues present their meta-analysis of the results of more than 30 studies on the association between exposure to tobacco smoke from parental smoking during pregnancy and cancer in childhood [*EHP* 108:73–82].

The biological plausibility of the association between passive tobacco smoke and childhood cancer is based on studies that show that children do take in tobacco components—including carcinogens and mutagens—when exposed to tobacco smoke during both gestation and childhood. Activation of procarcinogens in human fetal and placental tissues has been demonstrated, as has smoke-induced damage to DNA in human placenta.

In conducting their meta-analysis, the authors searched the medical literature for epidemiological studies on childhood cancer where smoking by one or both parents was recorded. The authors extracted from these studies characteristics of study design and results on risk from exposure to maternal or paternal smoking during pregnancy. They also extracted quantitative results, expressed as numbers of cigarettes per day smoked by the parents, where those data were available. For neoplasms for which risk estimates were available from at least three different studies, the authors combined the relative risk (RR) for any exposure to tobacco smoke into a meta-analysis based on a random effects model.



A meta-analysis of studies, though limited, indicates increased risk of cancer in children exposed to tobacco smoke from parental smoking.

The results of the meta-analysis suggest an increased risk following exposure to maternal tobacco smoke on the order of 10% (RR = 1.10). The authors state that this increased risk is small and is not clearly concentrated in any specific neoplasm. The only neoplasm for which a significant association was found was leukemia, but the authors say that increase could be explained by information bias (inaccurate recall) and confounding factors such as drug and chemical exposures, parental occupational exposures, diet, and socioeconomic status.

Fewer studies addressed exposure to tobacco smoke from the father. Using those that were available, the authors found a RR of 2.08 for non-Hodgkin lymphoma, 1.17 for acute lymphocytic leukemia, and 1.22 for central nervous system tumors or brain cancer.

The authors report that the overall interpretation of the studies reviewed was hampered by the crude exposure assessment used. Most of the studies did not include quantitative exposure variables, and those that had that information did not provide evidence of a dose–response relationship. They conclude that further studies are needed to overcome the practical difficulties of identifying adequate numbers of cases of childhood cancer and the possible limitations of the available epidemiological investigations.

–John S. Manuel

Catch the Drift

Assessing Risk from Pesticide Spraying

Farm workers are regularly exposed to pesticides in the course of their labors, and the effects on their health have long been a source of concern. Less well understood and more difficult to study is the risk to residents in nearby communities who are exposed to pesticides that drift when crops are sprayed from airplanes or trucks. In this month's issue, Mary Ward and colleagues have determined that one viable approach to identifying people potentially exposed to agricultural pesticides is to combine analysis of historical records with the technologies of remote sensing and geographic information systems (GIS) [*EHP* 108:5–12].

Many rural residents live within a few hundred yards of fields where pesticides are sprayed. In the past, studies primarily focused on pesticide applicators and exposure was estimated by using questionnaires or biological monitoring. Questionnaires have limitations in that respondents may be unaware of the types of agricultural pesticides in use near their homes. Biological monitoring is useful in estimating past exposure to pesticides with long half-lives but not exposure to the active ingredients and by-products of many pesticides that have short half-lives. Another method—measuring pesticide concentrations in household dust—may provide a useful approach for determining historical exposure, but interpreting the data requires knowledge about the proximity of residences to crop fields and pesticide levels in homes that has not been well defined. Providing some of this information is where sensing and mapping technologies may help.